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Wittmann, Lutz ; Zehnder, Daniel ; Jenni, Oskar G ; Landolt, Markus A

Abstract: **BACKGROUND:** Sleep onset and maintenance problems are a frequent complaint after traumatic events in children. However, the association of traumatic experiences and disturbed sleep remains to be explained. **OBJECTIVE:** To examine the incidence of sleep onset and maintenance problems in children after road traffic accidents and identify potential predictors of sleep onset and maintenance problems, including putative psychopathological mechanisms as well as stressors affecting the family system. **METHOD:** In 33 children treated for injuries after road traffic accidents, sleep and measures of psychopathology were assessed 10 days, 2 months, and 6 months after hospital admission. The predictive value of four clusters of predictor variables for children's sleep onset and maintenance problems was prospectively tested by multiple regression analyses. These clusters included socio-demographic, injury- and accident-related, and psychopathological variable clusters as well as factors reflecting stressors concerning mothers and family. **RESULTS:** Children suffering from posttraumatic stress reported a prolonged subjective sleep latency. The severity of sleep onset and maintenance problems was predicted by female sex and the child's as well as mothers' posttraumatic stress disorder (PTSD) severity. **CONCLUSIONS:** Sleep onset and maintenance problems in children after trauma appear to result from a complex interaction of multiple factors. Our findings support the transactional model of sleep-wake regulation that bears implications for the development of adequate intervention strategies.

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Predictors of children's sleep onset and maintenance problems after road traffic accidents

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Background: Sleep onset and maintenance problems are a frequent complaint after traumatic events in children. However, the association of traumatic experiences and disturbed sleep remains to be explained.

Objective: To examine the incidence of sleep onset and maintenance problems in children after road traffic accidents and identify potential predictors of sleep onset and maintenance problems, including putative psychopathological mechanisms as well as stressors affecting the family system.

Method: In 33 children treated for injuries after road traffic accidents, sleep and measures of psychopathology were assessed 10 days, 2 months, and 6 months after hospital admission. The predictive value of four clusters of predictor variables for children's sleep onset and maintenance problems was prospectively tested by multiple regression analyses. These clusters included socio-demographic, injury- and accident-related, and psychopathological variable clusters as well as factors reflecting stressors concerning mothers and family.

Results: Children suffering from posttraumatic stress reported a prolonged subjective sleep latency. The severity of sleep onset and maintenance problems was predicted by female sex and the child's as well as mothers' posttraumatic stress disorder (PTSD) severity.

Conclusions: Sleep onset and maintenance problems in children after trauma appear to result from a complex interaction of multiple factors. Our findings support the transactional model of sleep-wake regulation that bears implications for the development of adequate intervention strategies.

Keywords: *Posttraumatic stress disorder; sleep; children; road traffic accidents*

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Substantial proportions of children experience psychopathological sequelae such as acute or post-traumatic stress disorder (ASD/PTSD; American Psychiatric Association [APA], 2000) after potentially traumatic events. For instance, based on their recent review of 12 empirical studies, Olofsson, Bunketorp, and Andersson (2009) estimated a PTSD prevalence of 27% in children and adolescents one to two months after sustaining injuries due to road traffic accidents (RTA). A diagnosis of PTSD includes symptoms of re-experiencing

the traumatic event (cluster B) as well as less trauma-specific symptoms such as avoidance and hyperarousal (clusters C and D). Among the most common non-specific consequences of stress and trauma are disruptions of sleep (Sadeh, 1996). Adverse effects of childhood trauma on sleep have been found immediately (Pynoos et al., 1987) as well as years after trauma (Glod, Teicher, Hartman, & Harakal, 1997) and can still be demonstrated in adulthood (Bader & Schäfer, 2007). At the same time, sleep problems appear to hamper recovery

from traumatic events and to facilitate adverse psychopathological outcomes (Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2010).

Several hypotheses account for the pathways by which posttraumatic stress may affect sleep. Bader and Schäfer (2007) summarized evidence linking nocturnal worry and rumination to insomnia after trauma. With recurring memories such as unwanted thoughts about the event, the first symptom of the PTSD re-experiencing cluster partially covers this type of cognitive hyperactivation. Sleep disorders following trauma may be a result of hypervigilance. For instance, Moore (1989) assumed that interpersonal trauma prevents development of a safe attachment style, resulting in the conviction that one needs to stay alert and be on guard at all times instead of sleeping. From a biological perspective, posttraumatic stress may be associated with elevated levels of physiological arousal that are incompatible with conditions necessary for sleep onset. For instance, Germain, Buysse, and Nofzinger (2008) assume that “hyperactivity of the amygdala and attenuated activity of the medial prefrontal cortex [...] maintain or increase activity in arousal-promoting brain centers, and reduce activity in sleep-promoting centers. The resulting pattern of persistent arousal could directly contribute to complaints of insomnia” (p. 191). Potential pharmacological mechanisms of stress-induced changes in sleep have been summarized by Pawlyk, Morrison, Ross, and Brennan (2008). The only etiological factor explicitly mentioned by DSM-IV (APA, 2000) are posttraumatic nightmares (Wittmann, Schredl, & Kramer, 2006). The distressing emotions of nightmares not only disrupt sleep. Nightmares may also trigger or increase fear of going to sleep, which may already be present, particularly in younger children. Finally, Sadeh and colleagues (Sadeh, 1996; Sadeh & Anders, 1993) emphasized that not only stressors and children's psychopathological reactions are of relevance in this context. In their transactional model of sleep-wake regulation (Sadeh & Anders, 1993), they suggest additional consideration of variables such as parental psychopathology or stressors affecting the family system.

This perspective is supported by studies demonstrating effects of marital stability and family conflicts (Gregory, Caspi, Moffitt, & Poulton, 2006; Mannering, Harold, & Leve, 2011) as well as maternal separation anxiety (Scher, 2008) and depression (Karraker & Young, 2007; Stoleru, Nottelmann, Belmont, & Ronsaville, 1997) on children's sleep problems. However, to date, no empirically confirmed theory conclusively explains the association of traumatic experiences and disturbed sleep (Bader & Schäfer, 2007).

Objective

The aims of this study were twofold: (1) to examine the incidence of sleep onset and maintenance problems in

children after road traffic accidents; (2) to identify potential predictors of sleep onset and maintenance problems including the reviewed putative psychopathological mechanisms (e.g., nightmares, intrusions, and hypervigilance) as well as stressors affecting the family system.

Methods

Participants

For the purpose of the present study, an interview on sleep behaviour was added to the psychometric test battery of an already ongoing study. This randomized controlled trial (RCT; Zehnder, Meuli, & Landolt, 2010) tested the effectiveness of a single-session early psychological intervention for children after RTA. During the recruitment period (September 2004 to September 2007), families of children consecutively admitted to the University Children's Hospital Zurich were asked to participate. Inclusion criteria were RTA-related medical in- or outpatient treatment, age ranging from 7 to 16 years, sufficient fluency in German, as well as absence of severe head injury (Glasgow Coma Scale (Teasdale & Jennett, 1974) score >9) and no previous evidence of intellectual impairment. Interviews about sleep were added to the test battery in June 2006. Longitudinal data on sleep variables were available for 33 of the 99 participants of the original RCT.

Procedure

The local Ethics Committee approved the study. If children agreed to participate, their parents were asked for written informed consent. Assessments were performed 10.5 ($SD = 2.6$) days (t0/baseline assessment), 2 months (78.3 days, $SD = 18.7$, t1) and 6 months (197.3 days, $SD = 16.7$, t2) after the child's accident. Trained graduate psychologists using standardized interviews assessed the children. Children's interviews took 30–45 min and were performed in the child's home (13 interviews (13.1%) were performed in hospital). Additionally, medical variables were obtained from the hospital files. Data on the mothers were obtained via questionnaires. Families received 50 Swiss Francs as compensation for completion of all three assessments. Half of the children participated in a short intervention (ca. 30 min) immediately after the baseline assessment. This intervention comprised a detailed reconstruction of the accident, identification of accident-related appraisals, and psychoeducation about possible stress reactions. Possible coping strategies regarding how to deal with stress reactions were discussed with the children and their parents. Additionally, a leaflet including information about posttraumatic stress and a contact address was handed out (for details see Zehnder et al., 2010). Assessments were performed 2 and 6 months post accident by different interviewers

blind to the children's treatment status. Neither sleep variables nor posttraumatic stress were significantly related to group status (intervention vs. control group) at any time. For instance, neither sleep onset and maintenance problems (t1: ($t(31) = -0.09$, $p=0.93$), t2: ($t(31) = 0.97$, $p=0.34$)) nor subjective sleep latency (t1: ($t(31) = 1.14$, $p=0.27$), t2: ($t(31) = 0.06$, $p=0.96$)) nor PTSD severity (t1: ($t(23.5) = 0.54$, $p=0.60$, unequal variances), t2: ($t(23.3) = 0.39$, $p=0.70$, unequal variances)) were related to group status. Therefore, data from the intervention and control groups were collapsed into the same analyses for the present study.

Measures

ASD and PTSD

ASD was assessed using the Interview for Acute Stress Disorder (Interview zur Akuten Belastungsstörung, IBS-A-K, J; Steil & Fuchsel, 2005). PTSD was assessed with the Clinician-Administered PTSD Scale, Child and Adolescent Version (CAPS-CA; Nader et al., 2002; German version: Steil & Fuchsel, 2005). Both instruments apply the same structure by assessing each respective symptom with a frequency and an intensity rating ranging from "0" to "4". To establish a diagnosis of ASD or PTSD according to DSM-IV-TR (APA, 2000), a symptom was considered as present if the frequency of the experience received a score of at least "1" and the intensity rating at least "2". Subsyndromal ASD/PTSD was diagnosed according to Bryant, Salmon, Sinclair, and Davidson (2007) if criteria for one of the symptom clusters were not fulfilled. Previous studies have confirmed the reliability and validity of these instruments (Nader et al., 2002; Steil & Fuchsel, 2005). Symptom severity was assessed by ASD/PTSD total scores (sum of frequency and intensity ratings) which showed excellent internal consistencies (Cronbach's α ranging from 0.88 to 0.95 at the different assessments). For analysis of the relation between PTSD and sleep onset and maintenance problems, the PTSD total score was calculated without consideration of the sleep (D1) and nightmare (B2) items.

Depression

Depressive symptoms were assessed by the Children's Depression Inventory (CDI; Kovacs, 1985; German version: Depressionsinventar fuer Kinder und Jugendliche (DIKJ); Stiensmeier-Pelster, Schürmann, & Duda, 2000). A total severity score ranging from 0 to 52 can be obtained by summing the 26 items (3-point Likert scales from 0 to 2). The total scores were converted to T-scores based on a representative German norm population (Stiensmeier-Pelster et al., 2000). A cut-off T-score of 60 has reliably been shown to identify children with clinically relevant depression (Stiensmeier-Pelster

et al., 2000). In this study Cronbach's α ranged from 0.88 to 0.91 at the different assessment. As T-values rather than raw values are applied, it was not possible to calculate a total severity score without consideration of the DIKJ sleep item (No. 15). However, DIKJ raw total scores with and without the sleep item were almost perfectly correlated at all three assessment points (all $r > 0.995$). Thus, only marginal effects of diagnostic overlap are to be expected for analyses of the relation between depression and sleep onset and maintenance problems.

Sleep

At 2 and 6 months post accident assessment, children were asked to report their usual bedtime (lights off time) and time of awakening for both weekdays and weekends. Then, children indicated how they usually wake up (spontaneously by themselves/alarm clock/family member) and estimated their subjective sleep latency (how many minutes they needed to fall asleep after lights off, without distinguishing between weekdays and weekends). Adapting the format of CAPS-CA item D1, four separate items were additionally constructed assessing frequency and intensity of sleep onset and sleep maintenance problems: "Did you usually have problems falling asleep?" ("0" = "never", "4" = "daily or almost every day"); "If so: how much of a problem did you have falling asleep?" ("0" = "no problem", "4" = "very big problem"); "Did you usually wake up during the night?" ("0" = "never", "4" = "daily or almost every day"); "If so: how much of a problem did you have with waking up during the night?" ("0" = "no problem", "4" = "very big problem"). These four items were summed up to result in a sleep onset and maintenance problems score whose internal consistency (Cronbach's α) was 0.84 at t1 and 0.76 at t2. The CAPS-CA scoring rule for symptoms (frequency ≥ 1 and intensity ≥ 2) was applied to rate if sleep onset and/or sleep maintenance problems were present.

Nightmares

At 2 months post accident assessment, children were asked to rate how often they had experienced nightmares since the accident on a five point Likert scale ranging from "0" = never and "1" = rarely to "4" = most of the time.

Injury severity

Injury severity was assessed by a physician applying the Modified Injury Severity Scale (MISS), a reliable and widely accepted measure (Mayer, Matlak, Johnson, & Walker, 1980). The MISS assesses the severity of injuries in different bodily systems. MISS score ranges from 1 to 75; scores above 25 indicate a severe injury.

Dangerousness of accident

At t0, children rated on a three point Likert scale how dangerous the accident had been (“0”=rather not dangerous; “1”=moderately dangerous; “2”=rather dangerous).

Socio-economic status

At t0, paternal occupation and maternal education were gathered from mothers using a 6 point Likert scale. Summing up these scores, a socio-economic status (SES) score ranging from 2 to 12 was obtained. Based on their SES score, children’s families were allocated to one of the following three classes: lower class (SES 2–5), middle class (SES 6–8), or upper class (SES 9–12). In a previous study this measure proved to be a reliable and valid indicator of socio-economic status in Switzerland (Landolt, Vollrath, & Ribi, 2002).

Life events

Occurrence of 12 major life events (e.g., change of residence, unemployment in the family, or parental separation) experienced by the family during the 12 months period preceding the accident was assessed 10 days post accident based on the mothers’ reports. A life event score was calculated representing the number of the family’s life events. Children’s index accidents were not included in this score.

Psychotherapy and medication

At baseline assessment, children’s mothers were asked if the child had received any psychotherapeutic or psychiatric treatment prior to the accident and if the child was currently receiving any medication. At t1 and t2, mothers were asked if the child had received any psychotherapeutic or psychiatric treatment since the accident.

PTSD symptom severity of mothers

At t1, maternal symptoms of posttraumatic stress disorder were assessed by the validated German version of the Posttraumatic Diagnostic Scale (PDS; Griesel, Wessa, & Flor, 2006). The PDS is one of the most frequently used self-rating scales in trauma research. We applied part B rating the frequency of the 17 PTSD symptoms (criteria B–D) during the past months on a 4-point scale (ranging from “0”=not at all or only one time to “3”=five or more times a week/almost always). A sum score ranging from 0 to 51 reflects the PTSD symptom severity. At t1, Cronbach’s α for PDS total score of mothers’ was 0.86. Additionally, it was noted if mothers witnessed or were involved in their children’s accidents.

Family support

The Family Relationship Index (FRI) is a 27-item questionnaire consisting of three subscales of the Family Environment Scale (Moos & Moos, 1994; Schneewind,

Beckmann, & Hecht-Jackl, 1985). With expressiveness, cohesion, and conflict, three areas of family functioning are assessed. The resulting sum score is the most commonly applied measure of family support, with numerous studies supporting its reliability and construct validity. However, in this study Cronbach’s α was only 0.55.

Data analysis

Statistical analyses were performed with IBM SPSS statistics 19 (SPSS Inc., Chicago, IL, USA). Time in bed was calculated as difference between going to bed and getting up. Sleep duration was calculated as difference between time in bed and subjective sleep latency. Effect sizes (Cohen’s d) rather than group comparisons were calculated for differences in sleep parameters between children with vs. without a full or subsyndromal diagnosis of PTSD at t1 due to the small number of children receiving a PTSD diagnosis. The influence of four different clusters of predictor variables as assessed at t0 or t1 on later sleep onset and maintenance problems (sum score) at t2 was tested by separated multiple linear regression analyses (predictors were entered simultaneously). The first two clusters represent socio-demographic and accident/injury/treatment related factors. The third cluster comprises variables reflecting children’s psychopathology including PTSD severity (without nightmare and sleep item), nightmares (as explicitly mentioned as causal factors of sleep problems by DSM IV), depression severity, and psychotherapy during the study period. In case PTSD severity would significantly predict later sleep onset and maintenance problems, post hoc general linear models controlling for sleep onset and maintenance problems at t1 tested for specific effects of presence of recurring memories (as an indicator for rumination) and hypervigilance on sleep onset and maintenance problems at t2. The fourth cluster included stressors affecting the family system as life events during last 12 months, mothers’ PTSD severity, and family support. Sleep onset and maintenance problems at t1 were controlled for by entering them as a further predictor in each regression model. The decision to calculate regression models for four separated clusters of up to five predictors rather than testing all potential predictors within one model is related to the limited test power due to a small sample size. Variables not meeting the distributional characteristics required for parametric testing as indicated by Kolmogorov-Smirnov tests were handled according to Tabachnick and Fidell (1996) as follows: dichotomization for frequency of nightmares since the accident at t1 (never vs. rarely–most of the time), number of life events during the 12-month period preceding the accident (0 vs. ≥ 1) and dangerousness of accident (rather not dangerous/moderately dangerous vs. rather dangerous); log transformation for PTSD scores of

children and mothers (both t1); inverse transformation for depression T-values of children (t1). After transformation, the latter three variables met assumptions of normality.

Three missing values for PTSD severity of mothers at t1 and one missing value for a child's subjective sleep latency at t1 were imputed applying an expectation-maximization algorithm (PRELIS) based on information from other assessment time points. Furthermore, no information was available on psychotherapy during the study period for one child. Results of the regression analyses on psychopathological variables of children virtually did not differ if this missing value was replaced with "yes" or with "no". As the majority of children did not receive psychotherapy during the study period, this missing value was replaced with "no psychotherapy" to maintain this participant for the regression model.

Results

Sociodemographic and accident related variables

Twelve participants (36.4%) were females; mean age of the 33 children was 11.8 ($SD=2.4$, $range=7-15$) years. Sixteen children (48.5%) perceived their accidents as "rather dangerous". Mean injury severity (MISS) was 6.0 ($SD=4.2$, $range=1-21$). Table 1 summarizes further sociodemographic and accident related variables.

Table 1. Sociodemographic, accident and injury related characteristics of 33 children

Variable	<i>n</i>	%
Female sex	12	36.4
Living with both biological parents	22	66.7
Socio-economic status		
Lower class	3	9.1
Middle class	11	33.3
Upper class	17	51.5
Unknown	2	6.1
No previous road traffic accidents	31	93.9
Unknown	1	3.0
Type of road traffic accident		
Pedestrian	12	36.4
Bicycle	9	27.3
Motorbike	3	9.1
Car passenger	6	18.2
Other	3	9.1
Mild traumatic brain injury (Glasgow Coma Scale score 10–15)	4	12.1
Surgery performed	9	27.3
Hospitalization	20	60.6

Psychopathology

According to mothers' reports, five children (15.2%, no information available for one child) had received psychotherapeutic treatment prior to the accident. Four children (12.1%, no information available for one child) received additional psychotherapy during the study period. Seven children (21.2%, no information available for one child) received medication at the time of the baseline assessment. ASD (10 days post accident) and PTSD (2/6 months post accident) total scores were 22.1 ($SD=18.9$), 18.8 ($SD=19.9$), and 13.5 ($SD=18.7$), respectively. ASD and PTSD scores were not significantly related to sex (t0: $t(17.0)=-1.46$, $p=0.16$, unequal variances; t1: $t(16.5)=-1.53$, $p=0.15$, unequal variances; t2: $t(18.7)=-1.18$, $p=0.25$, unequal variances) or age (t0: $r=0.11$, $p=0.56$; t1: $r=-0.02$, $p=0.93$; t2: $r=0.11$, $p=0.56$). Depression (DIKJ) T-scores were 48.1 ($SD=10.2$) at t0, 45.2 ($SD=10.0$) at t1, and 43.7 ($SD=10.2$) at t2. All children fulfilled the objective as well as subjective stressor criterion of ASD/PTSD diagnosis. Ten days post accident, one (3.0%) participant was diagnosed with full ASD, a further six (18.2%) with subsyndromal ASD. Rates of children diagnosed with full/subsyndromal PTSD were 2 (6.1%)/2 (6.1%) at 2 months and 2 (6.1%)/1 (3.0%) at 6 months post accident. Rates of children exceeding cut-off value on the depression self-rating were 4 (12.1%, t0), 4 (12.1%, t1), and 3 (9.1%, t2). In summary, eight children (24.2%) can be considered to suffer from clinically relevant psychopathological reactions at some point during the 6 months post accident period. Thirteen (39.4%) children reported having experienced nightmares since the accident at t1. The proportions of girls (41.7%) and boys (38.1%) experiencing nightmares at t1 did not differ significantly (Fisher's exact test, ns).

Maternal/familial stressors

Mean family support sum score, as assessed by the Family Relation Index, was 11.0 ($SD=3.2$, $n=29$). Mean PDS total score of mothers was 4.7 ($SD=5.5$, $n=30$). Receiving scores above 10, five mothers (16.7%) qualified for moderate to severe symptomatology (Griesel et al., 2006). Six mothers (18.2%) had witnessed their child's accident without being involved themselves. Witnessing the accident was not related to PTSD-severity in mothers ($t(28)=0.09$, $p=0.93$). However, mothers' and children's PTSD severity scores were significantly correlated at t1 ($r=0.47$, $p<0.01$, $n=30$). According to mothers' reports, families had experienced a mean of 1.2 ($SD=1.4$, $range=0-5$, no information available for two families) life events during the year preceding the accident. Nineteen families (57.6%, no information available for two families) had experienced at least one event.

Sleep variables

Going to bed and getting up time as well as resulting time in bed (weekdays and weekends) at t1 are presented in Table 2 separately for children with and without a diagnosis of full or subsyndromal PTSD.

For the 29 children without a PTSD diagnosis, all sleep parameter are virtually identical with data reported for a sample of 522 twelve-year old Belgian children (Spruyt, O'Brien, Cluydts, Verleye, & Ferri, 2005). For instance, weekdays' going to bed and getting up time in both study groups are 21.1 h and 9.4 h, respectively. Also, sleep onset latency reported for the Belgian sample (25 min for weekdays and 20 min for weekends) is virtually identical with the subjective sleep latency (22.6 min without differentiation between weekdays and weekends) of our 29 children without PTSD. Therefore, data for the 29 children without PTSD diagnosis of our group of subjects can be considered as reflecting healthy normal sleep. However, strong effect sizes were detected in our data for sleep duration comparing children with and without PTSD-diagnosis, especially during weekdays. Obviously, this difference is mostly explained by large differences in subjective sleep latency. Children with a full or subsyndromal PTSD diagnosis needed on average more than three times longer to fall asleep compared to children without diagnosis. During weekdays, time in bed was 0.6 hours shorter for children with PTSD due to an earlier waking up time. However, as these children reported family members or alarm clocks awakened them, this finding cannot be interpreted as indicating a sleep maintenance problem. Accordingly, time in bed at weekends virtually did not differ between the two groups. Mean sum scores for sleep onset and maintenance problems were 2.9 ($SD=3.8$) at t1 and 2.0 ($SD=2.0$) at t2. Five children

(15.2%) reported sleep onset problems at t1, as did four children (12.1%) at t2. Additionally, two children (6.1%) reported sleep maintenance problems at t1, as did one child (3.0%) at t2.

Prediction of sleep onset and maintenance problems at t2

Initial sleep onset and maintenance problems at t1 were entered as control variables in all four regression analyses, each time significantly predicting later sleep onset and maintenance problems at t2. The first multiple regression analysis included sex, age, and living with both biological parents as sociodemographic predictors of sleep onset and maintenance problems at t2 (adjusted $R^2=0.35$, $F(4)=5.20$, $p<0.01$, $N=33$). Only female sex significantly predicted sleep onset and maintenance problems at t2 ($B=1.22$, $SEB=0.58$, $\beta=0.30$, $p<0.05$). A post-hoc comparison indicated that girls experienced significantly more sleep onset and maintenance problems at t2 than boys ($t(31)=-2.20$, $p<0.05$). In the second regression model on accident, injury, and treatment related predictors (adjusted $R^2=0.27$, $F(4)=3.97$, $p<0.05$, $N=33$), none of the predictors rating of the accident as dangerous, injury severity (MISS), or hospitalization reached significance. The third regression model tested the effects of psychopathology at t1 on later sleep onset and maintenance problems at t2 (adjusted $R^2=0.33$, $F(5)=4.11$, $p<0.01$, $N=33$). Of the predictors PTSD severity, nightmares, severity of depression and psychiatric or psychotherapeutic treatment since the accident, only children's PTSD symptom severity (without sleep and dream item) significantly predicted sleep onset and maintenance problems at t2 ($B=1.93$, $SEB=0.87$, $\beta=0.43$, $p<0.05$). Post hoc general linear models controlling for sleep onset and

Table 2. Sleep variables (interview data) for children with and without PTSD diagnosis 2 months post accident

Variable		PTSD – ($n=29$)		PTSD + ($n=4$)		<i>d</i>
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Weekdays	Going to bed time (h)	21.1	0.7	21.2	1.4	–0.1
	Getting up time (h)	6.9	0.5	6.3	0.7	1.0
	Time in bed (h)	9.8	0.9	9.1	1.5	0.6
	Sleep duration (h)	9.4	0.9	7.8	0.9	1.8
Weekends	Going to bed time (h)	22.6	1.1	23.0	1.8	–0.3
	Getting up time (h)	9.2	1.4	9.7	1.3	–0.4
	Time in bed (h)	10.6	1.2	10.7	0.7	–0.1
	Sleep duration (h)	10.2	1.1	9.5	0.4	0.9
	Subjective sleep latency (min)	22.6	19.3	75.0	38.7	–1.7

PTSD –/+ = participants without/with a diagnosis of full/subsyndromal posttraumatic stress disorder; sleep duration = time in bed – subjective sleep latency; *M* = mean; *SD* = standard deviation; *d* = Cohen's effect size for comparison PTSD– vs. PTSD+; h = hours; min = minutes; subjective sleep latency without differentiation between weekdays and weekends.

maintenance problems at t1 found no effect of presence of recurring memories ($F(1, 32) = 0.08, p = 0.78$) on later sleep onset and maintenance problems at t2, but an almost significant influence of presence of hypervigilance ($F(1, 32) = 4.05, p = 0.53$). The fourth regression model including stressors of the family system included family support, mothers' PTSD severity at t1, and presence of at least one life event during the last 12 months (adjusted $R^2 = 0.22, F(4) = 2.96, p < 0.05, N = 29$). Of these variables, only mothers' PTSD severity significantly predicted later sleep onset and maintenance problems in children ($B = 2.06, SEB = 1.00, \beta = 0.38, p < 0.05$). Due to missing values in mothers' self-ratings (PDS and FRI), this analysis was restricted to $n = 29$.

Discussion

This study observed a pronounced elevation of subjective sleep latency in children suffering from traumatic stress and identified female sex as well children's and mothers' PTSD severity as predictors of the detected sleep onset and maintenance problems.

All accidents reported by our participants met the DSM-IV criteria for traumatic events. Nevertheless, injury and treatment related variables illustrated that our participants experienced a broad range of accident severity. The rather low rate of ASD and PTSD diagnoses in our group of children after RTA is in line with results obtained from samples of adult Swiss accident victims (Schnyder, Wittmann, Friedrich-Perez, Hepp, & Moergeli, 2008). The rate of 16.7% of mothers qualifying for moderate to severe PTSD symptomatology most closely resembles the rate of 18.1% reported by Allenou et al. (2010) for a sample of 72 mothers 5 weeks after their child's motor vehicle accident. Interestingly, mothers' posttraumatic stress severity was not related to witnessing the accidents but to children's PTSD severity. Thus, mothers may be traumatized by the pathological condition of their children rather than by the accidents themselves.

Subjective sleep parameters of children without a diagnosis of PTSD at t1 most closely resembled those of a sample of 522 twelve-year old Belgian children (Spruyt et al., 2005). Thus, the sleep behaviour in our subjects appears to be typical for children from a Central European country. However, large effect sizes indicated that children suffering from PTSD get substantially less sleep. This appears to be due to prolonged subjective sleep latency (75.0 vs. 22.6 min), qualifying for sleep onset problems. This is line with results from the applied sleep items indicating that sleep onset problems were much more prominent compared to sleep maintenance problems. Our finding of an increased sleep latency in traumatized children replicates evidence based on actigraphic measurements in prepubertal

children with a history of physical or sexual abuse (Glod et al., 1997).

A regression analysis of socio-demographic predictors identified female participants as a group with elevated risk for sleep onset and maintenance problems following RTA. This is noteworthy as previous research on predictors of insomnia in adolescents revealed inconsistent results (Roberts, Roberts, & Chan, 2008). Accident and injury related variables did not predict later sleep onset and maintenance problems. This is in line with previous results indicating that injury severity and threat appraisal are not related to posttraumatic stress in children after RTA (Landolt, Vollrath, Timm, Gnehm, & Sennhauser, 2005). Incidence of nightmares was not significantly related to sleep onset and maintenance problems. This is surprising as nightmares are the only etiological factor for posttraumatic sleep problems explicitly mentioned by DSM-IV (APA, 2000) and a French study of a large representative sample found a relation between nightmares and abnormally long sleep onset as well as sleep with many awakenings (Ohayon, Morselli, & Guilleminault, 1997). Posttraumatic stress severity (without dream and sleep items) at t1 significantly predicted sleep onset and maintenance problems at t2. This supports hypotheses assuming that sleep onset or maintenance problems after trauma may be a function of posttraumatic stress levels. Post hoc GLMs controlling for initial sleep onset and maintenance problems specifically tested for the influence of two PTSD symptoms that have been identified in previous research to be related to sleep problems. The presence of recurrent memories did not predict later sleep onset and maintenance problems. However, future studies should apply more precise measures of rumination before a final conclusion is drawn on this putative etiological factor. As the presence of hypervigilance was almost significantly related to later sleep onset and maintenance problems ($p = 0.053$), future studies should test this promising concept with larger samples. Furthermore, future studies considering physiological stress markers in larger samples need to test the predictive value of different physiological (e.g., brain activity, cortisol levels) and psychological factors and their interaction. The finding that mothers' PTSD severity predicted sleep onset and maintenance problems in children adds a further facet to evidence indicating an influence of mothers psychopathology (e.g., Karraker & Young, 2007; Stoleru et al., 1997) on children's sleep. Thus, our results underscore the importance of a transactional conceptualization of problems in sleep-wake regulation (Sadeh & Anders, 1993). Perceiving stress in its mother may reinforce a child's worries and fears. Also, mothers suffering from PTSD symptoms such as avoidance of traumatic memory triggers, emotional numbing, or irritability, may feel unable to provide their children the context necessary for elaboration of

traumatic memories or to help them to soothe themselves at bed time.

This study is not without limitations that need to be kept in mind for any interpretation of our results. First of all, our sample was rather small, limiting test power for the applied multivariate analyses in particular. The low levels of posttraumatic stress—although in line with other Swiss studies on accidental injuries—may impede comparability with more symptomatic samples. The applied test battery lacked objective/psychophysiological measures of sleep and stress. The relation between subjective and objective assessment of sleep parameters is complex (Wittmann et al., 2006). However, an ecological study performing polysomnographic measurements in the home rather than in the laboratory setting (Germain, Hall, Katherine Shear, Nofzinger, & Buysse, 2006) can be interpreted as supporting the validity of subjective sleep complaints in traumatized individuals. Given the finding that female sex and mothers' PTSD severity predicted sleep onset and maintenance problems in children, future studies should also assess paternal stress levels in order to detect a possible transgenerational interaction of the variables sex, sleep, and stress after trauma. Furthermore, future studies should consider putative factors as parental bonding, trauma history, premorbid personality, enuresis, anxiety, other sleep disorders (e.g., hypersomnia) and pre-existing sleep disorders.

Conclusions

Notwithstanding these limitations, our findings illustrate the role of sleep onset problems in children after RTA and emphasize the need to consider a broad range of etiological variables for this symptom. Given the adverse effects of chronically disturbed sleep on psychopathological outcomes after trauma (Bryant et al., 2010), the need to effectively address insomnia in traumatized children is obvious. Thus, it needs to be assured that clinicians are aware of sleep problems in childhood trauma victims. A large range of sleep diagnostic instruments is available (Spruyt & Gozal, 2011) and should be regularly applied. If sleep problems are present, existing approaches for the treatment of traumatized children could be complemented by tailored psychotherapeutic and pharmacological interventions (Mindell et al., 2006; Mindell, Kuhn, Lewin, Meltzer, & Sadeh, 2006). However, our findings also emphasize the importance of systemic rather than individual only approaches in the assessment and treatment of children who are potentially suffering from posttraumatic stress and sleep problems. Encouraging results for such an approach come from a study showing that a family-centered intervention significantly improved sleep problems in a group of 33 infants (Skuladottir & Thome, 2003).

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There is no conflict of interest in the present study for any of the authors

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